Posttraumatic Stress Disorder Symptoms, Physiological Reactivity, Alcohol Problems, and Aggression Among Military Veterans

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This study examined the association between posttraumatic stress disorder (PTSD) symptomatology and aggressive behavior among a sample of male Vietnam veterans ($N = 1,328$). Results indicated that the hyperarousal PTSD symptom cluster evidenced the strongest positive association with aggression at the bivariate level when compared with the other PTSD symptom clusters. When the PTSD symptom clusters were examined together as predictors, hyperarousal symptoms evidenced a significant positive relationship with aggression, and avoidance/numbing symptoms were negatively associated with aggression. Examination of potential mediators indicated that hyperarousal symptoms were directly associated with aggression and indirectly related to aggression via alcohol problems. Reexperiencing symptoms were associated with aggression only indirectly and through their positive association with physiological reactivity and negative association with alcohol problems. Study results highlight the complexity of the relationship between PTSD symptoms and aggression, and suggest possible mechanisms explaining this association.

Keywords: posttraumatic stress disorder, aggression, physiological reactivity, alcohol, hyperarousal

Anger and aggression are common manifestations of military-related trauma (Novaco & Chemtob, 1998). Aggressive behavior alienates family members and negatively impacts social support networks (Matsakis, 1991) and may be associated with a variety of physical health problems (Miller, Smith, Turner, Guijarro, & Hallet, 1996). To date, we have very little understanding of the processes through which military-related trauma may lead to aggressive behavior. Research in this area may contribute to the development of more effective clinical interventions designed to target aggressive behavior among this population (Chemtob, Novaco, Hamada, & Gross, 1997).

Studies have demonstrated strong associations between posttraumatic stress disorder (PTSD) symptoms and measures of aggression (Byrne & Riggs, 1996; McFall, Fontana, Raskind, & Rosenheck, 1999). Further, relative to those without the disorder, male veterans with PTSD evidence higher rates of violent outbursts and aggressive behavior, express more hostility, and exhibit poorer anger control (Beckham, Feldman, Kirby, Hertzberg, & Moore, 1997; Carroll, Rueger, Foy, & Donahoe, 1985; Jordan et al., 1992; Kulka et al., 1990; Lasko, Gurvits, Kuhne, Orr, & Pitman, 1994; McFall et al., 1999). In their study of help-seeking male veterans, Beckham et al. (1997) found that approximately 75% of those with PTSD had engaged in physical aggression over the previous year, and those men reported an average of 22 physically aggressive acts over that time. In contrast, physical aggression was reported by 17% of the non-PTSD veterans, with an average of less than one aggressive act during the year.

Recent evidence indicates that a focus on PTSD as a singular construct may obscure differential relationships among its separate symptom clusters. In particular, the hyperarousal cluster may have a unique predictive role and has been found to be a strong prospective predictor of other components of the posttraumatic response (Schell, Marshall, & Jaycox, 2004). This cluster is charac-

terized by anger difficulties and other affective, physiological, and cognitive symptoms related to increased arousal (sleep and concentration problems, hypervigilance, exaggerated startle response; American Psychiatric Association, 1994). Zillmann's (1971) excitation-transfer theory suggests that perceived physiological arousal intensifies angry experiences and increases aggressive behavior when attributed to a provocative situation. Although most of the evidence in support of this theory is based on experimental studies conducted among normative populations (see Rule & Nesdale, 1976), individual-differences research among nonnormative samples has also demonstrated that aggression is related to both objectively measured physiological arousal (see Lorber, 2004) and perceived arousal (e.g., Margolin, John, & Gleberman, 1988). Thus, one might expect the hyperarousal cluster to exhibit an especially strong association with aggression. L. A. King and King (2004) found that only hyperarousal symptoms were associated with intimate partner aggression when considered together with PTSD emotional numbing symptoms in a study of a national sample of Vietnam veterans. This finding has yet to be replicated, and we are not aware of any published study that has examined the associations between the separate PTSD symptom clusters and general (nonrelationship) aggressive behavior.

Other variables that may partially account for the relationship between PTSD symptoms and aggression are beginning to be identified. Several laboratory studies of military veterans have found PTSD to be associated with heightened physiological reactivity to trauma reminders (Buckley & Kaloupek, 2001; Keane et al., 1998). Physiological reactivity may serve as a pathway for the effects of hyperarousal symptoms on aggressive behavior, given that heightened noncued arousal may predispose individuals with PTSD to exhibit a greater physiological response during trauma-cued conditions, as well as research findings documenting a link between noncued hyperarousal symptoms and reactivity to trauma cues (Pfefferbaum et al., 2006). A recent meta-analysis among civilian samples indicated that heightened physiological reactivity in response to a range of negatively valenced experimental stimuli is positively associated with aggressive behavior (Lorber, 2004). Evidence further suggests that the presence of threat may strengthen this relationship (Smith & Gallo, 1999; Zillmann & Cantor, 1976). The hypothesized mediational role of reactivity to trauma cues in the current investigation is consistent with the excitation-transfer theory (Zillmann, 1971), as well as the proposition that among combat veterans, heightened reactivity in response to perceived threat leads to reduced ability to engage in self-monitoring or other inhibitory processes that otherwise restrain the expression of aggressive behavior (Chemtob, Hamada, Roitblat, & Muraoka, 1994; Chemtob et al., 1997; Novaco & Chemtob, 1998).

PTSD is also highly comorbid with alcohol problems across a range of trauma groups, and evidence supports the notion that PTSD symptoms typically precede alcohol problems (Stewart, 1996). The *self-medication hypothesis* (Khantzian, 1985) is often used to explain this association, whereby in the case of PTSD, alcohol is used to reduce the distress and heightened anxiety that accompany PTSD. Hyperarousal symptoms in particular are thought to lead to self-medication attempts (Stewart, 1996; Ullman, Filipas, Townsend, & Starzynski, 2005), and PTSD-positive individuals with alcohol dependence exhibit more hyperarousal symptoms than do those with cocaine dependence (Saladin, Brady,

Dansky, & Kilpatrick, 1995). Problematic alcohol use has been consistently implicated as a robust risk factor for aggression across a range of civilian (Brookoff, O'Brien, Cook, Thompson, & Williams, 1997; Murdoch, Pihl, & Ross, 1990; Murphy, O'Farrell, Fals-Stewart, & Feehan, 2001) and veteran samples (Savarese, Suvak, King, & King, 2001; Windle, 1994). A study of male veterans in alcohol rehabilitation found a 39% prevalence rate of self-reported partner assault over the previous year, with 20% of the veterans reporting severe assault (Gondolf & Foster, 1991). Thus, alcohol problems were hypothesized to serve as an additional mediator of the relationship between hyperarousal symptoms and aggression.

In the current study, we used a structural equation modeling (SEM) framework to examine the unique associations between the separate PTSD symptom clusters, physiological reactivity upon exposure to trauma cues, alcohol problems, and aggressive behavior among a sample of male military veterans receiving services at the Department of Veterans Affairs (DVA). We hypothesized that among the separate PTSD symptom clusters, hyperarousal would evidence the strongest relative positive association with aggressive behavior (accounting for the effects of the other PTSD symptom clusters), and physiological reactivity and alcohol problems were predicted to partially account for this association.

Method

Data Source and Sample

Participants were male veterans who participated in a multisite trial initiated under the auspices of the Cooperative Studies Program of the DVA that examined the utility of psychophysiological measures in predicting the presence or absence of PTSD (see Keane et al., 1998). All participants had served in the Vietnam theater of operations between August 1964 and May 1975. Other key inclusion criteria were as follows: (a) The veteran was currently using inpatient or outpatient DVA services, and (b) the veteran was not taking any autonomically active medication (e.g., beta blockers) and did not have any medical condition (e.g., cardiovascular disease) that might significantly alter his physiological responding (see Keane et al., 1998, for full details).

Recruitment of veterans involved in DVA programs in psychiatry, substance abuse, PTSD, and readjustment counseling took place over a period of 42 months between 1989 and 1992. Participants were drawn from 2,115 veterans who were nonconsecutively screened for eligibility at 15 DVA medical centers across the United States. The 1,461 individuals who met eligibility criteria were offered the opportunity to read and sign a study-wide consent form approved by the Institutional Review Board at that medical center. Of those who signed the form, 1,328 completed the initial nonpsychophysiological study assessment and constitute the sample for the present investigation. Psychophysiological testing was completed by 1,210 participants, but 42 of these individuals were eliminated owing to artifact or other technical issues, leaving data from 1,168 participants for the psychophysiological analyses.

Sixty-seven percent of study participants identified themselves as Caucasian, 20% as African American, 9% as Hispanic, 2% as American Indian/Alaskan Native, and 2% as Asian/Pacific Islander. On average, veterans were 43.2 years old ($SD = 3.8$ years) and had 13.9 years ($SD = 2.4$ years) of formal education. Partic-

ipants reported earning a mean of $17,194 ($SD$ = $17,502) per year. Over half (52%) of the participants were married at the time of the study. Veterans represented various branches of the military; specifically, 62% served in the Army, 23% served in the Marines, 9% served in the Navy, and 6% served in the Air Force. On average, participants reported "moderate" combat exposure, with a mean score of 18.9 (SD = 6.8) on the Combat Exposure Scale (Keane et al., 1989). Further sample details are available in Keane et al. (1998).

Diagnostic and Psychometric Measures

PTSD symptoms were measured using the PTSD module of the Structured Clinical Interview for the *DSM–III–R* (SCID; Spitzer, Williams, Gibbon, & First, 1989). The SCID is a semistructured diagnostic interview with good sensitivity and specificity (see Watson, 1990, for a review). Keane et al. (1998) found the measure to exhibit adequate test–retest reliability and interrater reliability across study sites. For the current project, all of the SCID PTSD symptoms were assessed. That is, SCID PTSD skip-outs were not used. In response to the 17 items, each representing one of the PTSD symptoms listed in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev. [*DSM–III–R*]; American Psychiatric Association, 1987), clinicians reported whether the symptom was *absent*, *subthreshold*, or *threshold* for the veteran during the past 6 months. Absent symptoms received a score of 1, subthreshold symptoms received a score of 2, and threshold symptoms received a score of 3. Ratings for symptoms composing the reexperiencing (Cluster B), avoidance/numbing (Cluster C), and hyperarousal (Cluster D) symptom clusters were summed to arrive at symptom cluster scores. Total SCID PTSD symptom scores averaged 36.18 (SD = 10.56) in the current study.

Alcohol problems were assessed using the CAGE questionnaire (Mayfield, McLeod, & Hall, 1974). The CAGE assesses feelings of guilt related to drinking, early morning drinking behavior, criticism received from others regarding drinking, and perceptions of the need to reduce drinking during the 6 months prior to assessment. A yes–no response was given for each question, and positively endorsed items were summed. Participants averaged a score of 1.38 (SD = 1.59) on these items. The CAGE has demonstrated good sensitivity and specificity for detecting alcohol problems among veterans (Liskow, Campbell, Nickel, & Powell, 1995). In the present study, the internal consistency reliability estimate for the CAGE was .87.

Aggression was examined using a six-item measure designed to tap both verbal aggression ("threatened someone with physical violence without a weapon"; "threatened someone with a weapon"; "was verbally abusive") and physical aggression ("destroyed property"; "had a physical fight with someone"; "used a weapon against someone"). This measure was significantly predicted by PTSD symptoms and depression in a recent investigation of a large sample of male combat veterans (Taft, Vogt, Marshall, Panuzio, & Niles, 2007), suggesting its construct validity among the population of interest. Responses were given on a 7-point scale ranging from 0 (*never*) to 6 (*more than 20 times*) and were referenced to aggressive incidents toward anyone in the 6 months prior to assessment. All items were recoded to reflect the number of aggressive behaviors that were positively endorsed. Scores derived from this computation method, known as *variety scores*,

have desirable psychometric properties and have been advocated for measuring aggression (Moffitt et al., 1997). This approach reduces skewness caused by a small number of high-rate offenders, limits error introduced in the estimation of aggression frequency, and is most defensible with respect to memory limitations regarding behavior frequencies. Participants endorsed an average of 1.71 types of aggressive behaviors (SD = 1.55) on this measure over the past 6 months.

Psychophysiological Procedure

The interview and questionnaire portion of the assessment was followed by a psychophysiological challenge task, administered by a study technician (see Keane et al., 1998, for full procedural detail). The procedure began with a 10-min resting baseline during which participants sat quietly. This was followed by a mental arithmetic task that served as a generic stressor. Following a 5-min rest–recovery period, participants were presented with a set of six standardized still images depicting outdoor scenes (neutral scenes) accompanied by a musical soundtrack. Another 5-min rest–recovery period preceded presentation of six standardized images of combat in Vietnam (combat scenes) accompanied by a matching soundtrack (e.g., helicopter sounds, gun shots). Content for the neutral scenes was selected to be distinct from Vietnam, whereas material for the combat scenes was intended to be representative of that war zone.

Another 5-min rest–recovery period preceded the imagery portion of challenge testing. This involved presentation of two standardized neutral scripts and two idiographic trauma scripts that had been composed by a study clinician working with each participant using semistructured methods outlined by Pitman, Orr, Forgue, de Jong, and Claiborn (1987). Neutral scripts were intended to be distinct from Vietnam, and trauma scripts depicted each participant's two most stressful combat-related experiences in Vietnam. Imagery scripts were presented in alternating order beginning with a neutral script. Each script presentation consisted of four sequential 30-s periods: (a) resting quietly; (b) listening to a prerecorded reading of the script; (c) vividly imagining the script content; and (d) resting quietly. The period between successive scripts was 1 min or the point at which a participant's heart rate returned to within 5% of its previous baseline value, whichever was longer.

Heart rate (HR) and skin conductance (SC) were two of the physiological measures recorded throughout the task periods. HR was recorded from 9-mm-diameter Ag/AgCl electrodes (Sensor-Medics, Yorba Linda, CA) filled with electrolyte paste and attached at standard lead I (arm) sites. HR signals were processed through a bioamplifier (Model S75-01) and cardiotachometer (Model S77-26; both from Coulbourn Instruments, Allentown, PA). SC was measured directly by an isolated skin conductance coupler (Model S71-23; Coulbourn Instruments) using a constant 0.5-V output through 9-mm-diameter Ag/AgCl electrodes filled with an isotonic paste. Electrodes were attached to the hypothenar surface of the nondominant hand, separated by 14 mm. Electronic signals were sampled twice per second, converted to digital values, and stored on a computer for postprocessing. This included procedures for artifact detection and removal, and summarization of recordings based on 30-s time blocks (e.g., each 1-min audiovisual presentation produced two 30-s mean values for each measure).

Reactivity scores for HR and SC levels during the audiovisual scenes were each computed by subtracting the overall mean of 30-s values for the neutral scene presentations from the corresponding overall mean for the combat scene presentations. Similarly, HR and SC reactivity scores for scripts were calculated by subtracting the mean of the two imagining periods for neutral scripts from the mean of the two imagining periods for combat scripts.

Analyses

Although the fourth edition of the *DSM* (*DSM–IV*; American Psychiatric Association, 1994) postulates an intercorrelated three-factor model for explaining the symptom structure of PTSD, there has been recent empirical support for intercorrelated four-factor models of PTSD (D. W. King, Leskin, King, & Weathers, 1998; Marshall, 2004; McWilliams, Cox, & Asmundson, 2005; Simms, Watson, & Doebbeling, 2002). Support has been found for two different four-factor PTSD symptom cluster solutions. Both models hypothesize that PTSD symptoms are explained by separate underlying latent factors that include reexperiencing, avoidance, and hyperarousal symptom clusters. However, the models diverge with regard to the fourth factor. The four-factor model originally confirmed by D. W. King and colleagues (1998) hypothesizes emotional numbing as the fourth factor, with this factor explaining symptoms of amnesia, diminished interest in activities, feelings of detachment from others, restricted range of affect, and sense of foreshortened future. Simms et al. (2002) found that their fourth factor explained the same symptoms noted by the D. W. King et al. (1998) model for emotional numbing, but with disturbed sleep, anger outbursts, and poor concentration as additional symptoms loading on this factor. The authors labeled this factor *dysphoria*.

Previous studies have used the Clinician-Administered PTSD Scale and self-report measures to compare three- and four-factor models of PTSD (D. W. King et al., 1998; Marshall, 2004; McWilliams et al., 2005; Simms et al., 2002). However, studies have yet to use the SCID (Spitzer et al., 1989) to compare the *DSM–IV* three-factor model with the D. W. King et al. (1998) and Simms et al. (2002) four-factor models. Thus, the current study used confirmatory factor analysis to compare and contrast the empirical support for each of these models using SCID interview data for assessing PTSD diagnostic symptoms. For the purpose of statistical comparison, a single-factor PTSD model solution was also tested and compared with the three- and four-factor models of PTSD. The model generating the strongest empirical support was to be used for testing the hypotheses of the current study.

SEM was used to test the hypothesized interrelationships among study variables. This methodology is especially useful for examining complex associations among multiple constructs. Measurement models were computed to test the adequacy of the hypothesized models in explaining the observed data. Structural models were then specified and evaluated in a stepwise manner to examine the direct and indirect effects of the different PTSD symptom clusters on aggression. Specifically, the first structural model tested the direct effects of PTSD symptom clusters on aggression, and the second structural model tested whether the effects of the PTSD symptom clusters were mediated by physiological reactivity and alcohol problems. Because parsimonious structural models are desirable, the strategy for achieving the final model solutions was

to trim the initial measurement and structural models of their nonsignificant pathways to produce a final structural model. To improve parsimony, we excluded nonsignificant pathways in the measurement model from the corresponding structural model solution. Initial structural models were then calculated, and nonsignificant pathways were trimmed to produce a final structural model solution.

For all SEM analyses, raw data were submitted to the Mplus program, Version 4.1 (L. K. Muthén & Muthén, 2006a). The full-information maximum likelihood estimator was used to compute all SEM solutions to accommodate missing data. To control for their effects, we included age and combat exposure in all analyses. Age and combat exposure were allowed to freely predict all other model variables. SCID PTSD symptoms were submitted to Mplus as ordered categorical data, given that these individual scale items had three ordered response options (i.e., absent, subthreshold, or threshold). CAGE alcohol items and aggression scale items were also submitted as categorical data, because Mplus treats items with dichotomous scores as categorical (L. K. Muthén & Muthén, 2006b). In addition, the total count scores from the aggression measure were submitted as ordered categorical data, given that the aggression variable was based on count scores. To account for the categorical nature of these data, Mplus bases model solutions on polychoric and tetrachoric correlations. SEM model estimators were based on the weighted least squares with mean and variance adjustment (WLSMV), as this estimator has been shown to be ideal for models involving categorical data (B. Muthén, du Toit, & Spisic, 1997).

Following previous research (Orcutt, King, & King, 2003; Street, King, King, & Riggs, 2003), aggression was conceptualized as being causally indicated by its scale total. In contrast to the effects indicator approach, in which observed variables are assumed to be effects of their corresponding latent construct, causal indicator modeling assumes that the observed variables cause the underlying latent variable (Bollen & Lennox, 1991; Edwards & Bagozzi, 2000). Causal indicators can be postulated in cases in which specific behaviors are hypothesized to define the construct of interest (e.g., number of behaviorally specific aggressive acts within the past 6 months). This method is consistent with Netland's (2005) conclusion that causal indicator modeling is more theoretically appropriate than effects indicator modeling when using behaviorally specific, events-based measures of violence, because these variables are the causal definition of the construct of aggression.[1]

Results

Reporting of Aggressive Behavior

Item-level aggression data indicate that participants reported the full range of behaviors included in the aggression measure (see Table 1). The most frequently endorsed item was "was verbally abusive" (65.4%), followed by "threatened someone with physical violence (without a weapon)" (42.2%). Approximately one quarter of the sample indicated that they had destroyed property (23.4%)

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[1] The effects indicator model yielded nearly identical results to the causal indicator model. Effects indicator model results are available from the authors by request.

Table 1
Endorsement of Aggressive Behaviors

Item	%
Threatened someone with physical violence (without a weapon)	42.2
Threatened someone with a weapon	11.5
Was verbally abusive	65.4
Had a physical fight with someone	24.3
Destroyed property	23.6
Used a weapon against someone	4.4

or had a physical fight with someone (24.3%). Aggressive behaviors involving weapons ("threatened someone with a weapon," "used a weapon against someone") were endorsed least frequently (11.5% and 4.4%, respectively).

Confirmatory Factor Analyses of PTSD Symptoms

Confirmatory factor analyses demonstrated stronger support for the *DSM–IV* (American Psychiatric Association, 1994) intercorrelated three-factor model in comparison with both the D. W. King et al. (1998) and Simms et al. (2002) intercorrelated four-factor models of PTSD. Although all three confirmatory factor analysis models demonstrated a significant model chi-square, this test has been shown to be extremely sensitive to even small model-data deviations with large samples and is, therefore, a poor measure of the overall statistical model (Tabachnick & Fidell, 2003). Another disadvantage of this test when using the WLSMV estimator is that the chi-square and *df* values are not calculated in the same standard manner as chi-square and *df* values associated with maximum likelihood solutions, which results in an unconventional interpretation of these statistics within the WLSMV method (L. K. Muthén & Muthén, 2006b; see Table 2 note for additional information). Therefore, the comparative fit index (CFI; Bentler, 1990), the Tucker–Lewis index (TLI; Tucker & Lewis, 1973), the root-mean-

square error of approximation (RMSEA), and the weighted root-mean-square residual (WRMR) are more desirable model fit indices. Turning to these fit indices, the one-factor model and the three-factor *DSM–IV* model both exceeded the minimum suggested value of .95 for the CFI, whereas the CFI for each of the four-factor models was slightly below this suggested cut score (Hu & Bentler, 1998). Both the one-factor model and the *DSM–IV* three-factor model fell below a suggested maximum value of .08 on the RMSEA, whereas the four-factor models did not meet this suggested model fit criterion (Browne & Cudeck, 1993; Hu & Bentler, 1998; Steiger, 1990). Finally, the *DSM–IV* model solution demonstrated the lowest comparative WRMR value, with lower values being more desirable (Tabachnick & Fidell, 2003). In summary, although all models demonstrated generally acceptable statistical fit, the *DSM–IV* three-factor model exhibited the best overall model fit indices (see Table 2).

Bivariate Associations Among Latent Study Variables

Prior to testing the simultaneous multivariate effects of PTSD variables on aggression, we examined bivariate associations among latent SEM variables (see Table 3). As expected, PTSD symptom clusters were highly correlated. Small to medium associations were found between the PTSD symptom clusters and physiological reactivity and alcohol problems. Associations between the reexperiencing and avoidance/numbing PTSD symptom clusters and aggression were in the medium to large range of magnitude, and the association between the hyperarousal cluster and aggression was in the large range. Statistical comparison of these bivariate relationships revealed a stronger positive association between hyperarousal and aggression than between reexperiencing and aggression, Steiger's $z(1054) = 2.23$, $p < .05$, and a stronger positive association between hyperarousal and aggression than between avoidance/numbing and aggression, Steiger's $z(1054) = 3.28$, $p = .001$. This finding suggested that at the bivariate level, hyperarousal had a stronger relationship with ag-

Table 2
Goodness-of-Fit Indices for WLSMV Measurement and Structural Models

Model	Fit index					
	χ^2	*df*	CFI	TLI	RMSEA	WRMR
PTSD confirmatory factor analysis						
One-factor model	503.96	77	.96	.99	.07	1.62
DSM-IV intercorrelated three-factor model	375.82	79	.97	.99	.05	1.32
King et al. (1998) intercorrelated four-factor model	676.96	66	.94	.98	.08	1.95
Simms et al. (2002) intercorrelated four-factor model	731.66	66	.93	.98	.09	2.00
Direct effects of PTSD on aggression						
Measurement model	505.79	126	.97	.99	.05	1.40
Final structural model	444.48	129	.96	.98	.05	1.34
Effect of PTSD on aggression via physiological reactivity						
Measurement model	974.99	137	.94	.98	.07	1.81
Final structural model	648.63	138	.94	.97	.06	1.58

Note. For all chi-square values, $p < .0001$. Chi-square and *df* values associated with the weighted-least-squares with mean and variance adjustment (WLSMV) method are not interpretable in the same conventional manner as chi-square and *df* values produced by the maximum likelihood estimator. When using the WLSMV estimator, the chi-square and *df* estimates are adjusted until a correct p value is obtained, thereby resulting in unconventional values. Formulas for estimating WLSMV chi-square and *df* values are reported in the Mplus technical appendices (Muthén, 1998-2004). CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root-mean-square error of approximation; WRMR = weighted root-mean-square residual; PTSD = posttraumatic stress disorder.

Table 3
Estimated Correlation Matrix of the Latent Study Variables

Variable	1	2	3	4	5	6
1. Reexperiencing symptoms	—					
2. Avoidance/numbing symptoms	.87*	—				
3. Hyperarousal symptoms	.91*	.91*	—			
4. Physiological reactivity	.31*	.27*	.28*	—		
5. Alcohol problems	.12*	.19*	.24*	.04	—	
6. Aggression	.42*	.38*	.50*	.18*	.27*	—

* $p < .05$.

gression relative to the other PTSD symptom clusters. Turning to the other bivariate correlations, associations between physiological reactivity and aggression and between alcohol problems and aggression were in the small to medium range of magnitude. Physiological reactivity and alcohol problems were essentially uncorrelated.

Direct Effects of PTSD Variables on Aggression

The direct effects measurement model had 4 latent and 20 observed variables. Age and combat exposure severity were observed variables. Reexperiencing, avoidance/numbing, and hyperarousal symptoms were identified by the SCID symptom items corresponding to the *DSM–IV* three-factor model of PTSD. Aggression was causally measured by the total variety score for aggressive acts.

An acceptable statistical fit was found for the measurement model that included age, combat exposure severity, the *DSM–IV* three-factor PTSD model, and aggression. Model fit indices suggested an adequately fitting model in that the TLI and CFI exceeded the minimum suggested value of .95 (see Table 2). In addition, the RMSEA was below the suggested maximum value of .08. Thus, the measurement model was shown to effectively account for the underlying factor data structure.

The structural model supported a direct association between the latent PTSD symptom variables and aggression (see Figure 1). As with the measurement model, the CFI, TLI, and RMSEA were within their acceptable respective ranges, suggesting an adequate fit for the overall hypothesized model (see Table 2). Regarding specific model pathways, age had a direct negative association with aggression and was indirectly associated with aggression through its negative associations with hyperarousal and avoidance symptoms (standardized total effects = −.22, $p < .001$; standardized total indirect effects = −.09, $p < .001$). The effects of combat exposure severity on aggression were entirely indirect via its positive relationship with hyperarousal and avoidance symptoms (standardized total indirect effects = .10, $p < .001$). Turning to the PTSD symptom clusters, significant direct effects of avoidance/numbing symptoms and hyperarousal symptoms were found. Specifically, higher avoidance/numbing symptoms were associated with lower levels of aggression. In contrast, hyperarousal symptoms were positively related to aggression. The pathway from reexperiencing symptoms to aggression was nonsignificant and

negative. In summary, hyperarousal was the only PTSD symptom cluster to exhibit a positive relationship to aggression. These results supported the hypothesis that hyperarousal symptoms would exhibit a stronger unique positive relationship with aggression versus the other PTSD symptom clusters.

Indirect Effects of PTSD Symptom Variables on Aggression Via Physiological Reactivity and Alcohol Problems

The indirect effects measurement model had 5 latent and 28 observed variables. As with the direct effects models, age and combat exposure severity were observed variables. Reexperiencing, avoidance/numbing, and hyperarousal were identified by the SCID symptom items corresponding to the *DSM–IV* three-factor model of PTSD. Latent physiological reactivity was indicated by observed HR and SC reactivity scores during visual scene and script-based presentations. The latent alcohol problems variable was indicated by the four CAGE items. Aggression was causally indicated by the total variety score for aggressive acts.

The measurement model supported the hypothesized latent variable structure (see Table 2). Specifically, although the CFI was just below the suggested cut-off of .95, the TLI was above .95 and the RMSEA was below .08. Taken together, these model fit indices suggested a generally acceptable model fit.

The structural model provided partial support for the hypothesis that hyperarousal symptoms would have indirect effects on aggression through physiological reactivity and alcohol problems (see Figure 2). The pathway between alcohol problems and reactivity was not calculated within the final structural model solution because this pathway was nonsignificant in the measurement models, despite being permitted to correlate. To improve model parsimony, we left this and other nonsignificant pathways out of the final structural model. The CFI and TLI exceeded .95 and the RMSEA fell below .08, suggesting an acceptable model fit to the data (see Table 2). Age had significant negative direct and indirect relationships with aggression (standardized total effects = −.22, $p < .001$; standardized total indirect effects = −.12, $p < .001$). The effects of combat exposure severity on aggression were entirely explained by its relationship with higher PTSD symptom cluster scores (standardized total indirect effects = −.09, $p < .001$). Regarding the PTSD symptom clusters, the effects of reexperiencing symptoms were entirely indirect in that reexperiencing symptoms were positively associated with physiological reactivity, which in turn was related to higher aggression (standardized indirect effect = .04, $p < .05$). In addition, reexperiencing symptoms were indirectly associated with aggression through their negative relationship with alcohol problems (standardized indirect effect = −.10, $p < .01$). Avoidance/numbing symptoms had a direct negative association with aggression, but their indirect effects were nonsignificant. Finally, hyperarousal was the only PTSD symptom cluster to have a positive direct association with aggression. Hyperarousal was also indirectly related to aggression through its positive relationship with alcohol problems (standardized indirect effects = .13, $p < .001$).

Discussion

The current study examined associations among the PTSD symptom clusters, physiological reactivity to trauma cues, alcohol
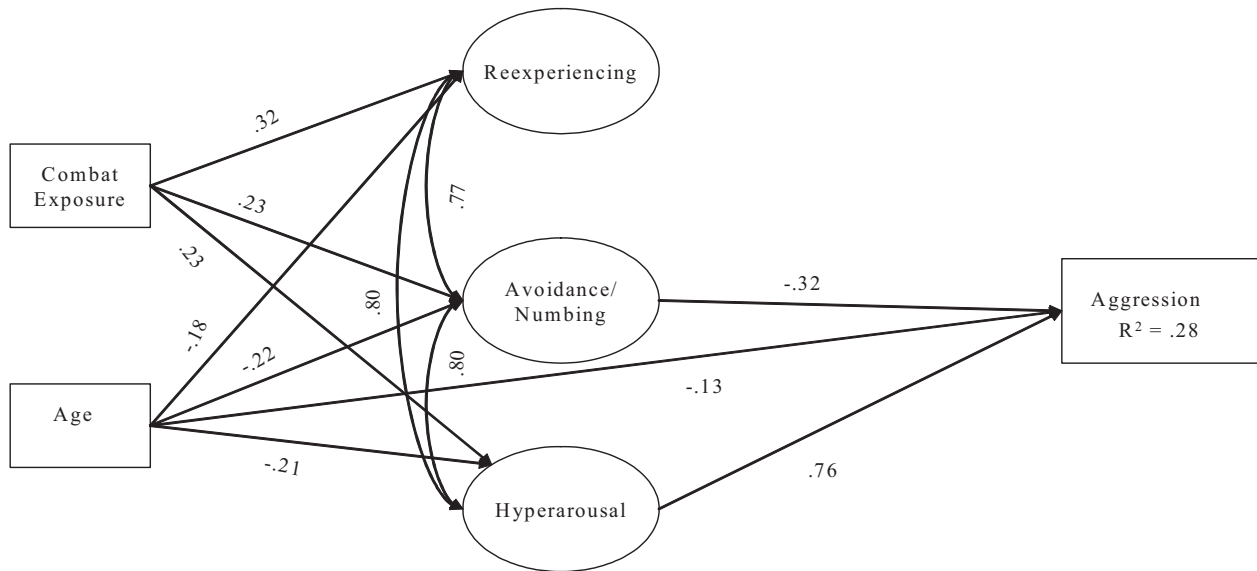
Figure 1. Standardized direct model of the latent posttraumatic stress disorder symptom variables in predicting latent aggression. All pathways shown are significant at $p < .05$. To improve model parsimony, we have excluded nonsignificant pathways in the initial model solution from the final structural model shown.

problems, and aggressive behavior among a large sample of male Vietnam veterans. The hyperarousal cluster evidenced a stronger positive association with general aggression at the bivariate level when compared with reexperiencing and avoidance/numbing and when the PTSD symptom clusters were considered together at the multivariate level, consistent with some prior research that has examined partner violence as the outcome (L. A. King & King, 2004). Taken together, these results suggest that the relationship between PTSD symptoms and aggression are best explained by higher hyperarousal cluster scores. In addition, hyperarousal symptoms were associated with a greater frequency of aggression through their relationship with alcohol problems. Findings for the mediational role played by alcohol problems are consistent with self-medication conceptualizations of PTSD-related alcohol problems that highlight the role of hyperarousal (Khantzian, 1985; Ullman et al., 2005), evidence suggesting heightened hyperarousal among those with alcohol problems (Saladin et al., 1995), and considerable evidence linking alcohol and aggression (e.g., Murphy et al., 2001).

Contrary to expectations, physiological reactivity did not mediate the effects of hyperarousal symptoms on aggression. A lack of an association between symptoms reflecting noncued arousal and cued physiological reactivity appears to be at odds with some previous work demonstrating this association (Pfefferbaum et al., 2006). However, research in this area is scant, and previous investigations have not tested the effects of the separate PTSD clusters and potential mediators simultaneously in structural equation models. Reexperiencing symptoms, on the other hand, did exert indirect positive effects on aggression through their relationship with physiological reactivity. These findings may reflect a link between self-reported physiological reactivity, a component of the reexperiencing symptom cluster (American Psychiatric Association, 1994), and reactivity to trauma cues in the laboratory. This is the first study that we are aware of to have demonstrated direct

associations between laboratory-based measures of physiological reactivity and aggressive behavior among a sample of military veterans, consistent with a large body of research among civilian samples (see Lorber, 2004).

An unexpected finding from the current study was the significant direct negative association between the PTSD avoidance/numbing cluster and aggression in the multivariate structural models. These results were in contrast to the positive relationship between PTSD avoidance/numbing and aggression at the bivariate level. The contrast in this pattern of results may be due to high degrees of multicollinearity among the PTSD symptom clusters, resulting in a small amount of nonshared residual variance of the PTSD symptom clusters in predicting aggression within the multivariate models. Because this negative relationship between avoidance/numbing symptoms and aggression in the SEM results may be due to the problem of multicollinearity with hyperarousal symptoms, this relationship should be interpreted with caution.

Symptoms of the hyperarousal cluster may lead to aggressive behavior owing to residual arousal being attributed to provocative situations, which increases anger and aggressive impulses (Zillmann, 1971). In a related manner, it is additionally possible that hyperarousal symptoms, as well as physiological reactivity and alcohol problems, lead to aggressive behavior owing to their relationships with impaired attention, cognitive processing, and inhibitory control (Eckhardt & Jamison, 2002; Gross, 1998; Thase, 2005). Research among civilians has suggested that excessive autonomic arousal interferes with higher order cognitive and self-regulatory processes (Patterson & Newman, 1993; Wallace, Newman, & Bachorowski, 1991), which may lead to impulsive and reactive aggression. Similarly, Chemtob and colleagues (Chemtob et al., 1994, 1997; Novaco & Chemtob, 1998) posit that among combat veterans, heightened physiological arousal in response to threat contributes to aggressive behavior owing in part to its effects on self-monitoring ability and other inhibitory processes. Further-
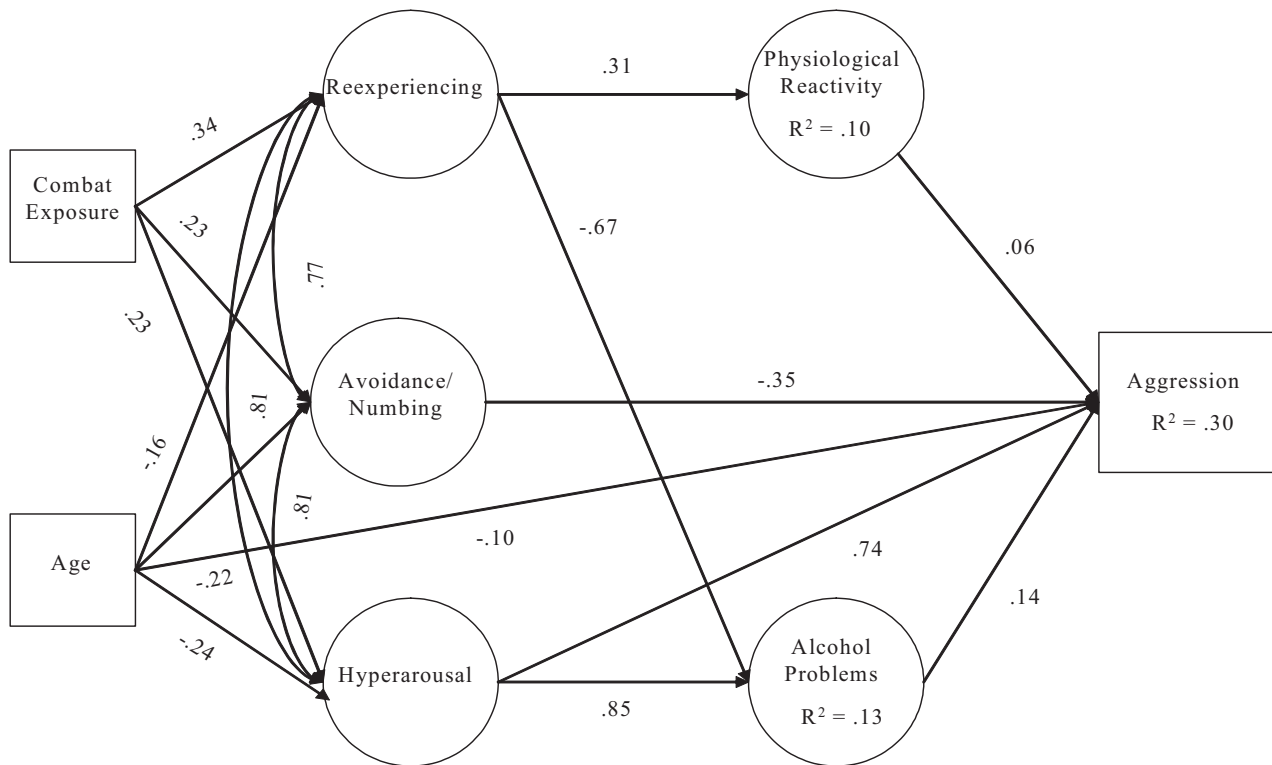
Figure 2. Standardized indirect model of the latent posttraumatic stress disorder symptom variables in predicting latent aggression via latent physiological reactivity and alcohol problems. All pathways shown are significant at $p < .05$. To improve model parsimony, we have excluded nonsignificant pathways in the initial model solution from the final structural model shown.

more, increased alcohol abuse may lead to aggressive behavior owing to diminished cognitive processing and inhibitory factors (Casbon, Curtin, Lang, & Patrick, 2003; Curtin & Fairchild, 2003; Curtin, Patrick, Lang, Cacioppo, & Birbaumer, 2001; Fillmore, Vogel-Sprott, & Gavrilescu, 1999; Marczinski & Fillmore, 2003), as well as a lowered threshold for behavior elicitation and potentiated psychomotor activity (Graham et al., 1998; Pihl, Peterson, & Lau, 1993). Although strong experimental work suggests that the proposed mechanisms of action may be operating, research explicitly examining such mechanistic models is greatly needed.

A more fine-grained analysis of different forms of aggression is warranted. Some evidence suggests potential differential relationships in the correlates of direct versus indirect forms of aggression (e.g., Archer, 2004). The limited item content for physical and psychological aggression in the current study did not allow us to conduct such an analysis. Future investigations that examine a broader array of aggressive behaviors are needed. The context of the aggression is also an important factor to consider in future research. For example, PTSD symptomatology has consistently been linked with intimate relationship abuse, and little work has elucidated mechanisms responsible for this association (Taft et al., 2005). Future research should more clearly assess whether aggression was perpetrated toward relationship partners or others and examine possible differences in the etiology of partner-specific versus general aggression. It is also not known the degree to which findings obtained from the current sample of Vietnam veterans

apply to veterans from other eras. In addition, the cross-sectional nature of this study precluded the ability to draw firm causal conclusions. Finally, it is important to note that although the PTSD measure used was based on outdated *DSM–III–R* criteria, the current study focused on PTSD symptoms, which were essentially unchanged in subsequent revisions. Changes in PTSD criteria have focused on revisions of the trauma criterion and the categorizing of the individual symptoms.

The current study highlights the need to better understand the relationship between PTSD symptoms and aggression in order to inform intervention efforts and ultimately ameliorate the numerous interpersonal and health consequences associated with such maladaptive behavior. Results suggest the potential value of treating hyperarousal symptoms in particular to reduce aggressive behavior displayed by male military veterans. In addition, anxiety reduction strategies that target trauma-related physiological reactivity and intervention for alcohol problems may be of value in reducing aggressive behavior in this population.

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